HEMIDIAPHRAGMATIC paralysis is a consequence of phrenic nerve injury. The most common causes are neck surgery, radiation, and malignancy. It may be a clinically silent entity, often discovered only when an elevated hemidiaphragm is found on a chest radiograph ordered for another indication. For this reason, especially in an era wherein preoperative chest radiographs are no longer routinely obtained, the prevalence of chronic hemidiaphragm paralysis in the surgical population is unknown, as is the incidence of complications related to this condition. We present a case of respiratory failure after laparoscopic cholecystectomy in a patient with preoperatively occult chronic hemidiaphragm paralysis.

Case Report

A 61-yr-old man was scheduled for laparoscopic cholecystectomy and open repair of a recurrent left inguinal hernia. His medical history was significant for mild hypertension. He had undergone a total thyroidectomy and radio ablation with I-131 in 1972 for thyroid cancer, with no evidence of recurrence. His only medication was l-thyroxine, 0.2 mg daily. There was no history of smoking or pulmonary disease. He stated that he walked one mile regularly and could climb two flights of stairs without becoming short of breath. He weighed 106 kg at a height of 173 cm. Pulse oximetry (SpO2) on the day of surgery revealed 97% saturation breathing room air in the semirecumbent position.

The patient was brought into the operating room, and anesthesia was uneventfully induced with midazolam, fentanyl, and sodium thiopental. Muscle relaxation was achieved with succinylcholine, and he was easily intubated by direct laryngoscopy. Anesthesia was maintained with intravenous fentanyl and isoflurane in a 45% oxygen in air mixture, and neuromuscular blockade was maintained with pancuronium. Vital signs were stable and unremarkable for the entire case. Expected changes in end-tidal CO2, airway pressures, and tidal volumes occurred with CO2 abdominal insufflation and returned to baseline with deflation. SpO2 was 100% throughout.

The surgical procedures were accomplished without complications, and after administration of neostigmine and glycopyrrolate, the gas mixture was changed to 100% oxygen, and the patient commenced spontaneous ventilation. However, despite apparently adequate ventilatory efforts, full train-of-four responses, and return of consciousness, SpO2 decreased to 89-91%. Multiple sigh breaths were administered to manage presumed atelectasis, without improvement. On auscultation, breath sounds were present bilaterally without wheezing. Airway pressures and respiratory system compliance were unchanged and normal. End-tidal CO2 readings were 45-50 mm Hg. Negative inspiratory force exceeded –25 cm H2O. Spontaneous minute ventilation was 10 l/min. The patient became fully awake and responsive, denying pain but admitting to some dyspnea. He was transported to the postanesthesia care unit (PACU) intubated and placed on ventilator support. Arterial blood gas (ABG) analysis on arrival revealed a pH, 7.29; PCO2, 49 mmHg; and PO2, 67 mmHg on 100% oxygen (FiO2, 1.0). A chest radiograph performed on admission was of suboptimal quality, but it appeared to demonstrate marked elevation of the right hemidiaphragm and diffuse densities in the left middle and upper lung. On ventilator support (FiO2, 1.0), the patient became more comfortable, and SpO2 increased to 100%. A second chest radiograph obtained 30 min after the first (fig 1) showed persistence of the hemidiaphragm elevation with improved aeration of the left lung. One hour later, ABG analysis (FiO2, 0.6) revealed pH, 7.33; PCO2, 43 mmHg; and PO2, 88 mmHg. The patient was admitted to the intensive care unit (ICU) and remained intubated for approximately 15 h. Before extubation (FiO2, 0.4), ABGs were pH, 7.40; PCO2, 40 mmHg; and PO2, 97 mmHg. Once extubated, the patient was weaned to room air, and on discharge from the ICU, SpO2 was 94%. His subsequent course was completely benign, and he was discharged on the second postoperative day. Frontal and lateral chest radiographs performed on the day of discharge revealed a persistently elevated right hemidiaphragm with modest basilar discoid atelectasis bilaterally. On reviewing the patient's records with his internist, it was determined that a hemidiaphragm elevation had been noted on previous chest radiographs, but fluoroscopy to document diaphragmatic paralysis had not been pursued.

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Fig. 1 Portable chest radiograph after 30 min of mechanical ventilation in the PACU. The right hemidiaphragm is markedly elevated, but left lung aeration appears normal.

Discussion

This case illustrates a previously unreported occurrence: respiratory failure in the setting of hemidiaphragm paralysis after laparoscopic cholecystectomy. In retrospect, this patient with a history of neck surgery and irradiation clearly had a chronically paralyzed and atrophic right hemidiaphragm, but was asymptomatic in his activities of daily living, including modest exercise. We hypothesize that insufflation of gas intraperitoneally may have caused significant differential upward displacement of the right hemidiaphragm with resultant atelectasis of that lung. This was not apparent intraoperatively as positive pressure ventilation maintained some degree of lung inflation. With the conversion to spontaneous ventilation, the negative airway pressure during inspiration then caused paradoxical upward motion of the right hemidiaphragm, seriously aggravating the atelectasis on that side, with resultant shunt and hypoxemia as was observed. Although modest desaturation on commencing spontaneous ventilation is not uncommon, it usually resolves quickly with assisted ventilation and 100% inspired oxygen. The magnitude and refractoriness of the desaturation in our patient alerted us to the presence of a problem and led us not to extubate in the operating room. A prolonged period of positive pressure ventilation with slow weaning to spontaneous ventilation was ultimately required to resolve the problem.

Laparoscopic cholecystectomy has been previously demonstrated in patients with normal diaphragms to result in significant impairment of diaphragmatic contractile function. However, impairment of diaphragmatic function after open cholecystectomy is also well documented. Based on this patient’s course, we speculate that laparoscopic procedures may present specific additional hazards in patients with hemidiaphragm dysfunction, but we cannot discount the possibility that the same events may have occurred after an open procedure.

Compensatory mechanisms allow patients with unilateral hemidiaphragm paralysis to be asymptomatic in activities of daily life and, whereas pulmonary function tests will reveal moderate abnormalities, nonathletically inclined patients may not observe any decrement in exercise tolerance. Patients with a history of neck surgery or irradiation should be carefully questioned to assess the possibility of phrenic nerve injury. Physical examination findings are subtle and consist of asymmetry in the chest examination to percussion, with absent breath sounds at the affected base. Thus historical and physical findings may escape recognition in the absence of initial suspicion. If a preoperative chest radiograph is available, the presence of hemidiaphragmatic elevation warrants evaluation to exclude hemidiaphragmatic paralysis before undertaking an upper abdominal procedure. Patients with known hemidiaphragm paralysis may be susceptible to unexpected desaturation on resumption of spontaneous breathing and should be extubated with caution. We invite reporting of any other cases of postoperative respiratory failure associated with hemidiaphragmatic paralysis.

References


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Intraoperative Immediate Diagnosis of Acute Obstruction of Tricuspid Valve and Pulmonary Embolism Due to Renal Cell Carcinoma with Transesophageal Echocardiography

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PATIENTS with the tumor thrombus in the inferior vena cava (IVC) have a great risk of pulmonary embolism and obstruction of tricuspid valve during operative management. There has been several reports about intraoperative cardiac arrest and death due to such situations. Therefore, immediate diagnosis and the subsequent management is an important challenge for anesthesiologists.

This case report describes a patient with renal cell carcinoma that extended into the IVC and caused acute obstruction of tricuspid valve and pulmonary tumor embolism intraoperatively, which were immediately diagnosed by transesophageal echocardiography (TEE) and were successfully managed by the rapid institution of cardiopulmonary bypass (CPB).

**Case Report**

A 55-yr-old woman, weighing 53 kg, was admitted to our hospital with appetite loss and left abdominal pain. Physical examination revealed a palpable mass in the left upper quadrant. Laboratory results showed a hematocrit of 30% and the elevation of leukocyte count (11,500), alkaline phosphatase (378 U/l), and lactic acid dehydrogenase (2,786 U/l). Radiologic examinations, including computed tomography, inferior venocavograms (fig. 1), and transsthoracic echocardiography revealed a left renal mass with a tumor thrombus extending upward to near the right atrium. The chest radiograph revealed the metastatic lesions in the left lower lung area. This patient

![Fig. 1. Inferior venocavograms demonstrating the tumor thrombus extending upward to near the right atrium (RA). The arrows indicate extension of the tumor thrombus in the inferior vena cava (IVC).](image-url)